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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	OCT 23	The Derwent World Patents Index suite of databases on STN has been enhanced and reloaded
NEWS	4	OCT 30	CHEMLIST enhanced with new search and display field
NEWS	5	NOV 03	JAPIO enhanced with IPC 8 features and functionality
NEWS	6	NOV 10	CA/CAplus F-Term thesaurus enhanced
NEWS	7	NOV 10	STN Express with Discover! free maintenance release Version 8.01c now available
NEWS	8	NOV 20	CA/CAplus to MARPAT accession number crossover limit increased to 50,000
NEWS	9	DEC 01	CAS REGISTRY updated with new ambiguity codes
NEWS	10	DEC 11	CAS REGISTRY chemical nomenclature enhanced
NEWS	11	DEC 14	WPIDS/WPINDEX/WPIX manual codes updated
NEWS	12	DEC 14	GBFULL and FRFULL enhanced with IPC 8 features and functionality
NEWS	13	DEC 18	CA/CAplus pre-1967 chemical substance index entries enhanced with preparation role
NEWS	14	DEC 18	CA/CAplus patent kind codes updated
NEWS	15	DEC 18	MARPAT to CA/CAplus accession number crossover limit increased to 50,000
NEWS	16	DEC 18	MEDLINE updated in preparation for 2007 reload
NEWS	17	DEC 27	CA/CAplus enhanced with more pre-1907 records
NEWS	18	JAN 08	CHEMLIST enhanced with New Zealand Inventory of Chemicals
NEWS	19	JAN 16	CA/CAplus Company Name Thesaurus enhanced and reloaded
NEWS	20	JAN 16	IPC version 2007.01 thesaurus available on STN
NEWS	21	JAN 16	WPIDS/WPINDEX/WPIX enhanced with IPC 8 reclassification data
NEWS	22	JAN 22	CA/CAplus updated with revised CAS roles
NEWS	23	JAN 22	CA/CAplus enhanced with patent applications from India
NEWS	24	JAN 29	PHAR reloaded with new search and display fields
NEWS	25	JAN 29	CAS Registry Number crossover limit increased to 300,000 in multiple databases
NEWS	26	FEB 13	CASREACT coverage to be extended
NEWS	27	Feb 15	PATDPASPC enhanced with Drug Approval numbers
NEWS	28	Feb 15	RUSSIAPAT enhanced with pre-1994 records
NEWS	29	Feb 23	KOREAPAT enhanced with IPC 8 features and functionality
NEWS	30	Feb 26	MEDLINE reloaded with enhancements
NEWS	31	Feb 26	EMBASE enhanced with Clinical Trial Number field
NEWS	32	Feb 26	TOXCENTER enhanced with reloaded MEDLINE
NEWS	33	Feb 26	IFICDB/IFIPAT/IFIUDB reloaded with enhancements
NEWS	34	Feb 26	CAS Registry Number crossover limit increased from 10,000 to 300,000 in multiple databases

NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

NEWS HOURS STN Operating Hours Plus Help Desk Availability

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 NEWS LOGIN     Welcome Banner and News Items
 NEWS IPC8      For general information regarding STN implementation of IPC 8
 NEWS X25       X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that
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FILE 'HOME' ENTERED AT 14:47:04 ON 05 MAR 2007

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                                              ENTRY    SESSION
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=> (autism or autistic) and (dipeptideyl peptidase or CD26 or CD 13 or CD69 or CD
69 or CD13 or CD 26)
L1             0 FILE AGRICOLA
L2             1 FILE BIOTECHNO
L3             0 FILE CONFSCI
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L4            0 FILE HEALSAFE
L5            0 FILE IMSDRUGCONF
L6            3 FILE LIFESCI
L7            0 FILE PASCAL

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L8            4 (AUTISM OR AUTISTIC) AND (DIPEPTIDEYL PEPTIDASE OR CD26 OR CD
                13 OR CD69 OR CD 69 OR CD13 OR CD 26)

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L9             3 DUP REM L8 (1 DUPLICATE REMOVED)

=> d l9 ibib abs total
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5/4/2004

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L9   ANSWER 1 OF 3  LIFESCI    COPYRIGHT 2007 CSA on STN
ACCESSION NUMBER:   2005:1197  LIFESCI
TITLE:              Heat Shock Protein and Gliadin Peptide Promote Development
                    of Peptidase Antibodies in Children with Autism
                    and Patients with Autoimmune Disease
AUTHOR:             Vojdani, A.*; Bazargan, M.; Vojdani, E.; Samadi, J.;
                    Nourian, A.A.; Eghbalieh, N.; Cooper, E.L.
CORPORATE SOURCE:   Section of Neuroimmunology, Immunosciences Lab., Inc., 8693
                    Wilshire Blvd., Suite 200, Beverly Hills, CA 90211; E-mail:
                    immunsci@ix.netcom.com
SOURCE:             Clinical and Diagnostic Laboratory Immunology [Clin. Diagn.
                    Lab. Immunol.], (20040500) vol. 11, no. 3, pp. 515-524.
                    ISSN: 1071-412X.
DOCUMENT TYPE:      Journal
FILE SEGMENT:       F
LANGUAGE:           English
SUMMARY LANGUAGE:   English
AB   Searching for a mechanism underlying autoimmunity in autism, we
     postulated that gliadin peptides, heat shock protein 60 (HSP-60), and
     streptokinase (SK) bind to different peptidases resulting in autoantibody
     production against these components. We assessed this hypothesis in
     patients with autism and in those with mixed connective tissue
     diseases. Associated with antigliadin and anti-HSP antibodies, children
     with autism and patients with autoimmune disease developed
     anti-dipeptidylpeptidase I (DPP I), anti-dipeptidylpeptidase IV (DPP IV
     (or CD26)) and anti-aminopeptidase N (CD13)
     autoantibodies. A significant percentage of autoimmune and
     autistic sera were associated with elevated immunoglobulin G
     (IgG), IgM, or IgA antibodies against three peptidases, gliadin, and
     HSP-60. These antibodies are specific, since immune absorption
     demonstrated that only specific antigens (e.g., DPP IV absorption of
     anti-DPP IV), significantly reduced IgG, IgM, and IgA antibody levels. For
     direct demonstration of SK, HSP- 60, and gliadin peptide binding to DPP
     IV, microtiter wells coated with DPP IV were reacted with SK, HSP-60, and
     gliadin. They were then reacted with anti-DPP IV or anti-SK, anti-HSP, and
     antigliadin antibodies. Adding SK, HSP-60, and gliadin peptides to DPP IV
     resulted in 27 to 43% inhibition of the DPP IV-anti- DPP IV reaction, but
     DPP IV-positive peptides caused 18 to 20% enhancement of antigen-antibody
     reactions. We propose that (i) superantigens (e.g., SK and HSP- 60) and
     dietary proteins (e.g., gliadin peptides) in individuals with predisposing
     HLA molecules bind to aminopeptidases and (ii) they induce autoantibodies
     to peptides and tissue antigens. Dysfunctional membrane peptidases and
     autoantibody production may result in neuroimmune dysregulation and
     autoimmunity.

L9   ANSWER 2 OF 3  LIFESCI    COPYRIGHT 2007 CSA on STN
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ACCESSION NUMBER: 2004:108019 LIFESCI
TITLE: Infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes are major instigators of autoimmunity in autism
AUTHOR: Vojdani, A.; Pangborn, J.B.; Vojdani, E.; Cooper, E.L.
CORPORATE SOURCE: 8693 Wilshire Blvd., Ste. 200, Beverly Hills, CA 90211, USA; E-mail: DrAri@msn.com
SOURCE: International Journal of Immunopathology and Pharmacology [Int. J. Immunopathol. Pharmacol.], (20031200) vol. 16, no. 3, pp. 189-199.
ISSN: 0394-6320.
DOCUMENT TYPE: Journal
FILE SEGMENT: F
LANGUAGE: English
SUMMARY LANGUAGE: English

1.131 / 1.132

AB Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of autism. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and ethyl mercury (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26). We assessed this hypothesis first by measuring IgG, IgM and IgA antibodies against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against ethyl mercury bound to human serum albumin in patients with autism. A significant percentage of children with autism developed anti-SK, anti-gliadin and casein peptides and anti-ethyl mercury antibodies, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These antibodies are synthesized as a result of SK, gliadin, casein and ethyl mercury binding to CD26 and CD69, indicating that they are specific. Immune absorption demonstrated that only specific antigens, like CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin, casein and ethyl mercury to CD26 or CD69, microtiter wells were coated with CD26 or CD69 alone or in combination with SK, gliadin, casein or ethyl mercury and then reacted with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these molecules to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-CD26 or anti-CD69 antibodies. The highest % binding of these antigens or peptides to CD26 or CD69 was attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosal (ethyl mercury) in individuals with pre-disposing HLA molecules, bind to CD26 or CD69 and induce antibodies against these molecules. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in autoimmune reaction in children with autism.

1.130 / 1.132

L9 ANSWER 3 OF 3 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 2003:36798209 BIOTECHNO
TITLE: Opioid peptides and dipeptidyl peptidase in autism
AUTHOR: Hunter L.C.; O'Hare A.; Herron W.J.; Fisher L.A.; Jones G.E.
CORPORATE SOURCE: A. O'Hare, Royal Hospital for Sick Children, Community Child Health Services, 10 Chalmers Crescent, Edinburgh EH9 1TS, United Kingdom.
E-mail: AO'Hare@ed.ac.uk
SOURCE: Developmental Medicine and Child Neurology, (01 FEB 2003), 45/2 (121-128), 40 reference(s)
CODEN: DMCNAW ISSN: 0012-1622

DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:36798209 BIOTECHNO
AB It has been hypothesized that autism results from an 'opioid peptide excess'. The aims of this study were to (1) confirm the presence of opioid peptides in the urine of children with autism and (2) determine whether dipeptidyl peptidase IV (DPPIV/CD26) is defective in children with autism. Opioid peptides were not detected in either the urine of children with autism (10 children; nine males, one female; age range 2 years 6 months to 10 years 1 month) or their siblings (10 children; seven males, three females; age range 2 years 3 months to 12 years 7 months) using liquid chromatography-ultraviolet-mass spectrometric analysis (LC-UV-MS). Plasma from 11 normally developing adults (25 years 5 months to 55 years 5 months) was also tested. The amount and activity of DPPIV in the plasma were quantified by an ELISA and DPPIV enzyme assay respectively; DPPIV was not found to be defective. The percentage of mononuclear cells expressing DPPIV (as CD26) was determined by flow cytometry. Children with autism had a significantly lower percentage of cells expressing CD5 and CD26, suggesting that they had lower T-cell numbers than their siblings. In conclusion, this study failed to replicate the findings of others and questions the validity of the opioid peptide excess theory for the cause of autism.